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HEMICRANIA AND ITS RELATIONS TO EPILEPSY AND HYSTERIA.*

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EVERY experienced practitioner knows that migraine, so common and dreaded, presents extremely diverse, clinical types. It is to be readily accepted that this variation in the type of manifestation is due to the different etiology of the cases.

Perhaps the reputed treatment of this disease becomes more successful, if its diverse etiological foundation is understood.

For a long time migraine was known as a constitutional, degenerative neurosis almost exclusively hereditary, which like other similarly conditioned nervous and physical diseases usually manifest themselves at biological periods of life and was described by Lieveing in 1874 and recently by Möbius in their excellent monographs.

The symptoms of this form of migraine, after a longer or shorter prodromal period, are general malaise, unilateral headache of special intensity, optic and acoustic hyperasthesia, nausea and even vomiting, anorexia, cessation of the attack after sleep usually. The face pale, rarely suffused during the attack.

As more rare symptoms of the attack and to be regarded as complications are to be mentioned initial temporary dimness of vision and scotoma (transition cases to hemicrania ophthalmica), olfactory hyperasthesia, ringing in the ears, vasospastic symptoms in the extremities (icy hands and feet). To the knowledge of this classical form of

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migraine is added that of the hemicrania opthalmica (Galezowski 1878, Fere and others), *i. e.*, the integrant attendance and domination of eye symptoms (*scotoma scintillans*) in the type of the attack of the hemicrania.

This variety is often that in which the hereditary constitutional form occurs. It may exist from the beginning as such or the migraine scotoma be developed during the course of the trouble.

Under all circumstances this form of migraine is the more severe.

So it is conceivable that here complicating brain diseases frequently occur, which are otherwise foreign to the type of simple migraine.

To be designated as such are unilateral parasthesias, motor and amnestic aphasia, transient hemipareses. Symptoms of irritation in hearing and taste, phenomena of agraphia, alexia, word-deafness, mental blindness (Berbez).

A further advancement in clinical knowledge was the fact, that migraine may have a *symptomatic* import. It has been found as an *acquired* disease originating late in life by reasons of conditions and changes, which lues cerebri, tumor cerebri (Lebert, Wernicke), the process of paresis (Sander, Mendel, Parinaud, Blocq, Charcot) and tabes (Oppenheim, Charcot, Roullet) have induced in the brain.

The tardive migraine thus appears in suspiciously ominous light, but it must not be overlooked, that, in rare cases at least, the hereditary constitutional migraine may first be developed late in life, possibly at the climacteric.

There are no absolutely certain signs of distinction, except hereditary taint and possibly familiar symptoms of the migraine.

It must be admitted, that the symptomatic migraine on organic basis is clinically presented as ophthalmic in the large majority of cases and *gradually* perfected with ophthalmic symptoms, not as in many cases of mild origin and significance. According to my experience very especially ominous appears to me the coincidence of hemiparasthesia in the symptom type of tardive hemicrania opthalmica. I have here never missed the early occurrence of progressive

paralysis or of organic focal disease (especially excephalomalacia).

But simple migraine, without any of these accompaniments, may also have this ominous significance, as I observed in a 16-year-old girl with progressive paralysis, afflicted with hereditary syphilis, yet without any family disposition to migraine. Consequently in the matter of diagnosis the chief weight should be placed on the absence of any family disposition to migraine.

In view of the fact of a possible symptomatic import of hemicrania the question arises, whether the attacks of this disease may not have a relation to certain neuroses (epilepsy, hysteria).

In regard to this question as to epilepsy Dejerine 1886 (l'hérédité dans les maladies du Systéme nerveux) and Féré 1890 (les épilepsies) have pointed to the extremely frequent occurence of epilepsy and hemicrania in the progenitors and relatives. Parry, Liveing, Gowers and others consider the two neuroses related and believe that they can pass from one to the other. Möbius refers to the fact that both are very often transmitted and homogeneously inherited, both usually begin in childhood, are manifested in attacks with a tendency to periodicity, often have the same exciting causes, that both may have the same prodromi, aura, polymorphism, incompleteness of the attacks, even status, pass into, respectively replace each other.

In both a permanent "alteration" in the brain must exist fundamentally.

Féré even went a step farther, in that he held epilepsy and migraine, particularly the ophthalmic, to be equivalent, manifestations of a *common* brain change.

Féré (*Revue de Med.* 1881 and "*Die Epilepsie*," German by Ebers 1896) declares eye migraine to be simply a "partial sensory epilepsy." This can be developed from simple migraine, but generally exists as such from the beginning. For years this sensory epilepsy may be limited to the eye symptoms (scotoma or hemianopsia, or both simultaneously). The symptoms of eye migraine could also be dissociated, in that the complemental migraine with vomiting follow a

transitory amblyopia or scotoma after a few days. Aphasia, paraesthesia of one side, hemiplegia may be accessory to the complete or incomplete eye migraine. But it may also happen that these symptoms occur paroxysmally in the same individual induced by the migaine. Epilepsy and especially its partial form is a frequent accompaniment of eye migraine.

The cases presented by Féré and others of epilepsy connected with such, respectively arising from them, are almost exclusively cases of ophthalmic hemicrania.

Gowers also calls attention to the fact, that in all his twelve cases of migraine transformed into epilepsy, the first had been attended by sensory aura.

In my experience also I do not know of a single case, where a simple migraine could be clinically related to epilepsy and consequently the occurrence of simple migraine and epilepsy in the same individual must be considered a mere coincidence, i.e. a complication, readily explainable by the great frequency of both neuroses of themselves and particularly in hereditary taint. The following cases chosen from many similar ones, is evidence of this assumption.

Case I. K., 19, laborer of a neuropathic family, not epileptic nor afflicted with migraine, somewhat feeble minded, has had frequent classical epileptic seizures since 13, which are not preceded by an aura. He is very irritable since his disease began and would kill himself on being refused the slightest wish. Therefore he was brought to the clinic. There it was ascertained that he had had attacks of migraine since 12, usually the most violent on the left side and recurring at intervals of a few weeks. Eye symptoms never occur in these attacks.

Féré's thesis on the epileptic import of (ophthalmic) migraine, requires according to my experience qualification and also amplification. In almost all cases within the scope of my observation where clinical relations between migraine and epilepsy were not to be proven, it is a matter of eye migraine, but on the other hand I have noted a large number of cases and followed them through a term of years, in which migraine opthalmica existed alone, but where no

epileptic foundation or only epileptic symptoms were to be proven.

Féré's view could only really exist, when the eye symptoms could be proven within the type of the migraine as something not belonging to it of itself but ascribed to epilepsy, then as a part of epilepsy within the migraine, which thus becomes an epileptic migraine. But prior experiences are insufficient, and it must remain an open question whether the eye migraine is clinically related to epilepsy in all cases. The clinical prognostic importance of the solution of this question is evident.

But that eye migraine *may* have such clinical relations, might be made clear by the following observations. On the other hand Féré's thesis needs amplification, in that those of an attack of sensory Jacksonian may have the same import as the eye symptoms in migraine.

In this rare combination of migraine the right to assign the sensory Jacksonian phenomena to epilepsy seems still greater than in the eye symptoms.

A preliminary question arises, namely: Whether the symptoms of an attack of sensory Jacksonian may be regarded as equivalent to those of a motor.

Facts, which I have observed in motor Jacksonian due to organic disease, in those attacks induced by hysteria, as well as in paretics, where sensory attacks may be observed as equivalent to the motor, justifies me in this assumption.

Pitres (*Revue de med.*, 1888, viii), offers the evidence, that not merely sensory, but even psychical attacks may replace the motor Jacksonian.

Oppenheim (*Handb. d. N-Krankheiten*, p. 437) claims that partial (Jacksonian) epilepsy may affect the sensory sphere, in that paraesthesia precedes the convulsion, accompany the twitchings, or "in that the paraesthesia is the only symptom of the irritable state, then forms in a certain measure an equivalent of the attack."

It is of no slight interest that in the large majority of cases of symptomatic migraine (tabes, general paresis, etc.) its form is the opthalmic or that accompanied by the symptomatic migraine, so that it might be attempted to recog-

nize the symptomatic migraine exclusively in these two forms. This point of view deserves being determined at any rate in the investigation of the clinical relations of migraine to epilepsy.

If it is attempted to go deeper into the question on such a basis, cases are offered, in which the same individual has in the same attack an epileptic and migraine seizure, *scotoma* appears as their common *aura*. This may besides acquire a direct import pointing to epilepsy, when it is of a red color. While the migraine always remains the same, the epileptic complimentary phase of the general attack may vary.

In the following nine cases three times it is a matter of classical epileptic seizures (cases 2, 3, 4), six times of sensory Jacksonian (cases 5-10). In one this introduces the general attack (10) so that in this single case, where no visual *aura*, *i. e.*, *scotoma* exists, it appears as though the paraesthesia (sensory *aura*) might be the representative of the *scotoma* of migraine.

In one of these cases (7) a state of post-epileptic confusion followed the attacks of sensory Jacksonian, at least ᵛ noteworthy fact in support of the theory of the significance of attacks of sensory Jacksonian as possible equivalents of motor, chiefly epileptic seizures.

Of no slight import is the fact, that some such epileptic seizures occur only at the time of attacks of hemicrania in the following casuistics, never as sporadic and isolated from the hemicrania.

The clinical connection then becomes undeniable and the above presumption that epileptic and hemicranial change or disposition in the brain are very closely related, is almost a certainty.

The observations, which justify such conclusions, are the following.

It is comprehensible that in all the most careful observation and examination, by means of the ophthalmoscope even, discover no focal symptoms, no signs of an organic brain disease, so that the objection to an organic symptomatic limitation to epileptic and hemicranial symptoms becomes untenable.

Case 2. Mrs. W., 43, whose mother was very nervous, neuropathic even, has had an habitual hemicrania since puberty. Climacteric about a year ago. The migraine has become very violent during the last four months and been associated with *scotoma*. During this time at the acme of the attack classical epileptic seizures.

Case 3. Mr. R., 34, of an apparently healthy family, unaffected by lues, *potus nimius*, *trauma capitis*, has had ophthalmic migraine since twenty years of age. When the attack is especially violent he has an epileptic seizure at its acme (loss of consciousness, general tonic-clonic convulsions, bites his tongue, etc.) Such attacks occur four times a year and are followed by malaise and mental apathy for twenty-four hours.

Case 4. Miss K., 18, has a mother and sister who have simple hemicrania.

The patient had convulsions at the age of 5, almost continual cephalaea since childhood, was a dull, stupid pupil and at 13 had attacks of right hemicrania (headache, vomiting,) which are preceded by black *scotoma* filling the greater part of the visual field. After a few months genuine epileptic seizures were associated with these attacks of hemicrania at their *acma* (loss of consciousness, suggilations, biting the tongue, etc.,) which recurred every few weeks, but never appeared as spontaneous paroxysms, *i. e.*, independently of the migraine.

In the meantime, and quite often the symptom complex of ophthalmic migraine occurred without being combined with epileptic phenomena.

She began to menstruate at 15, but puberty had no influence on either neurosis. Whereas from that time hysterical phenomena (*globus*, concentric contraction of the visual field, bilateral ovarian tenderness, etc.) presented themselves.

At 17 she was a frequent witness of the epileptic seizures of a boy. She then became very emotional and at 18 had Jacksonian epilepsy in the right upper extremity, which had no relation to the hemicrania, unattended by clouding of the consciousness, pupils not fixed, the seizures occurring

from once to five times a day and appeared like poor imitations of true Jacksonian attacks the patient had seen in the boy. Under bromides the attacks of epilepsy and ophthalmic migraine disappeared, while the hysterical imitations were unaffected.

Case 5. Miss S., 17, mother nervous and afflicted with hemicrania. She menstruated at 12, since then has had constitutional neurasthenia and an almost constant head pressure.

At 15 habitual migraine developed. Four times in the last two years it has been accompanied by hemianopsia and scotoma. Sometimes during these attacks, which are differentiated from the habitual menstrual, by their not occurring during menstruation, a sensory Jacksonian attack lasting an hour occurred on the right side of the face, tongue and hand.

Case 6. S., mechanic 58, apparently untainted, has had ophthalmic migraine since childhood. The attacks occur sixteen to thirty times a year and have increased in frequency of late. The attack begins with a dark spot in the visual field, followed by a headache on the same side. Rays of light are found at the periphery of these scotoma.

The migraine scotoma lasts about half an hour until the occurrence of the pain. The ophthalmic symptom occasionally consists of a white, glitttering C or Z, which appears first in one eye, then in the other, "as an undulating, light sparkling letter, which passes obliquely across the visual field." This letter is developed from a white speck.

At the acme of the pain then following the patient is giddy, unable to think, has a feeling of painful confusion and a "horrible" sensation, *i. e.*, he is conscious of a numbness in the face, tongue and upper extremity on the same side as the pain, is unable to speak and has the feeling of being paralyzed (sensory Jacksonian).

Case 7. Miss V., 18, menstruated at 15, apparently untainted, has had ophthalmic migraine since puberty, which at first lasted only an hour and a half, but of late four hours. At the acme of the attack, about half an hour

after the occurrence of the scotoma, sensory Jacksonian occurs (face, tongue, upper extremity), and on the same side as the pain. For a day after the attack the patient complains of vertigo, is forgetful, abstracted, confused, dazed, lets everything fall from the paraesthetic extremity, hence acts very awkwardly. She has only a summary memory of this post-migraine (post-epileptic?) stage.

In the interval she is perfectly well. Epileptic ancestors are wanting.

Case 8. Miss Z., mother has migraine, father irascible, several brothers and sisters have died of convulsions.

Menstruated at 14. *Commotio cerebri* at 18. Hemicrania began three months later. Attacks about every eight days.

Begin with scotoma before both eyes. These disappear in about twenty minutes. Then a feeling of numbness in the right upper extremity extending upward from the fingers. After about ten minutes the paraesthesia of the right upper extremity lessens, but now passes to the lower lip, tongue, so that the patient's speaking is affected. Headache now for the first, which is localized on the whole left side of the head. Nausea accompanies the whole attack. Vomiting rare.

Case 9. H. M., dress-maker, father nervous, irascible, who has "headache" a great deal. Her sister presents the same anomalies.

Patient has been of a nervous, excitable temperament since childhood, menstruated at 15 without special trouble.

The patient has had headache since nine years of age, which occurred twice a week, began during sleep, lasted all day, located in the left temple, when especially severe spread over the forehead to the right, accompanied by yawning and anorexia. At first they were attended by nausea and vomiting.

About the same age attacks of sensory Jacksonian with scotoma, acousma and aphasic symptoms occurred, which regularly passed into an attack of ordinary migraine.

This second category of attacks occur at intervals of three weeks to a month, without cause, suddenly, always

in the morning. The attack affects the right side of the body and lasts until it is transformed into the usual migraine on the left side in one half to one quarter of an hour. Without any premonitions the patient feels a "pricking of needles" in the right half of the tongue; in one to two minutes this "pricking" extends to the gums and the buccal mucous membrane of the right side of the mouth. Then a feeling of numbness and swelling occurs in the right half of both lips. After a few minutes the eye, ear and hand of the right side are simultaneously attacked. The right visual field seems filled with shining rays on a dark ground. She cannot see on the right side of the visual field, unless she turns her head to this side. While the scotoma disappears, roaring in the head, ringing in the right ear occurs. She does not hear her own voice; that of other people seems to come from a distance, as though her ear was stopped up. A feeling of stiffness and formication then occurs in the right upper extremity, which begins in the thumb, successively attacks the other fingers and extends from the finger tips to the elbow, possibly to the shoulder.

At this time the patient must talk slowly and at a great effort, because otherwise she confounds words or letters at least, *e. g.*, says "rütżt" for "nützt". It repeatedly happens that she cannot pronounce at first the names of common objects and finally compounds words falsely, *e. g.*, "knife-pen" for "pen-knife", which never occurred other than in the attack. She was painfully conscious of her aphasia and the incorrectness of her language.

Consciousness in such an attack is perfectly clear, the disposition depressed, emotional. The color of the face presents no change.

The patient is of good intelligence, somewhat anaemic, offers no signs of a derangement of the nervous system, except the deep reflexes are greatly exaggerated and marked tenderness in the region of the left ovary. Fundus normal. Residues in both tympani of repeated disease of the middle ear.

Case 10. September 29th 1896, L., 23, married,

book-keeper, asked protection of a policeman on the street from two men he fancied were following him, appeared mentally deranged and was taken to the psychiatric clinic (Vienna).

On admission the patient was disturbed, emotional, confused, afraid of those about, soon became quiet, went to sleep, and, after a few hours, awoke lucid. He only remembered that he had left the house about seven o'clock to go to the office, which he did not reach; of all that occurred until coming to himself at the clinic, the patient has absolute and permanent amnesia. His father, whose sister has epilepsy, is very nervous and excitable. During the first dentition period he had convulsions, also during typhoid while a boy. He was nervous and very irritable since childhood.

Five years ago an epileptic seizure in the theatre, probably from alcoholic excess, which was followed by a series of convulsions for three days. In June, 1896, another series after alcoholic excess. Since then frequent sporadic seizures in the evening.

For five years the patient has had an attack of petit mal every two or three weeks.

For some years the patient has had attacks of hemicrania, which are very readily induced by emotion, quickly disappear on the administration of antipyrine and on going to sleep. The attack begins with paleness of the face, paraesthesia (feeling of numbness) in the left hand, which lasts several minutes.

Pain then occurs in the right temple, which passes to the left temple and orbit and remains fixed. The left palpebral fissure becomes narrower and the patient optically and acustically hyperaesthetic. Scotoma and vomiting do not occur. Chronic paraesthesia of the left hand is accompanied by hesitating speech. Consciousness is unclouded.

In the interval the patient feels perfectly well and capable of work. Cranium and facial bones slightly asymmetrical. No signs of focal disease. Ophthalmoscopic examination negative.

In the following cases hemicranic and epileptic sym-

toms are dissociated, separated from each other in point of time, but united clinically by common eye symptoms, which however, may be regarded as the visual aura of an epileptic seizure or as migraine scotoma.

It may consist merely of the aura (abortive attack) or the "aura" pass into an attack of epilepsy or migraine. Isolated migraine with scotoma (visual aura) then evidently seems to be the equivalent of an epileptic seizure. On the other hand epilepsy, accompanied by a visual aura (11) and followed by post-epileptic vomiting and hemicrania (15) may be regarded as an equivalent of migraine.

The following six cases with invariable migraine symptoms show the polymorphism of epileptic phenomena. In case 15 confusion replaces the motor symptoms.

In this respect case 16 is especially interesting, in which at first only scotoma appearead, later epileptic seizures introduced by them. After temporary suppression of the epilepsy by attacks of scotoma with migraine, the epilepsy has recently reappeared, but in form of a physical equivalent accompanied by scotoma.

This temporary substitution of one symptom complex by another is a frequent occurrence in epilepsy.

This is interestingly shown in case 11, in 12 with respect to hemicrania.

Case 11. G.,17, school boy, of an apparently untainted, but tuberculous family, has had hemicrania, mostly on the left side, with a very brief initial scotoma since his tenth year.

At 16 classical epilepsy occurred from no apparent cause. His head always became hot, for a moment the whole visual field was filled with a dark red color and then he became unconscious. Occasionally a post-epileptic hallucinatory condition follows the convulsive stage, when he always sees a market place in Vienna, which at first is very large but always grows smaller until it seems a mere point. He sees twenty or more buildings in a row, which threaten to crush him, so that he is greatly frightened and crawls under the bed. Subsequently the whole affair seems like a dream.

Since the occurrence of the epilepsy the attacks of hemicrania have become much milder and more rare.

Case 12. Miss G., 40, cranium slightly rachitic. Mother had ophthalmatic hemicrania. The patient has had the same trouble since childhood. The migraine is preceded by a scotoma of short duration. Occasionally the headache does not follow it.

After intense emotion and influenza at 38 classical epilepsy occurred, which always began with visual aura (stars, sparks, scotoma). Since the advent of the epilepsy the attacks of migraine have become more rare.

Case 13. Miss L., 19, came under my treatment in September, 1889.

She is of a nervous family, but had never presented any neurotic symptoms previously, nor been seriously ill, menstruated at 15 without any attendant trouble, has never manifested any hysterical or epileptic symptoms, had been cheerful and enjoying life until 18, when she became very emotional over marriage complications, which were satisfactorily adjusted. After intense affect and a chill from sea bathing during menstruation six epileptic seizures in which she bit her tongue, occurred one night in August, 1888. Since then she has been nervous, irritable, emotional. Of late generally intensified seizures in November 1888, January, May, June, July, September 1889, which always ended in vomiting and were usually premenstrual. The attacks occurring previously as simple epilepsy acquire a peculiar clinical interest through the following statements of the patient. The beginning of each attack is the vision of a bright disk of the most diverse colors, occupying the left visual field exclusively. The disk often becomes smaller and then disappears. Then the attack does not occur. If the disk instead of getting smaller grows larger the attack occurs as soon as it fills the whole visual field.

There is no memory of these occurrences.

The findings of an eminent gynecologist were entirely negative. A healthy girl, no anaemia, no disease of the vegetative organs and absolutely no stigmata hysterica. No pain from pressure on the left supraorbital foramen, but the

immediate appearance of a golden circle, which disappears as soon as the pressure ceases. The patient is very anxious, evidently fearing a new attack. She states the disk is similar when an attack impends. She describes the circle as yellow outside and dark blue within.

Pressure over other portions of the trigeminus, on the bulb etc. does not cause these light phenomena.

Sodium bromide 3,0-4,0 pro die was ordered.

On May 18th, 1890, the patient gratefully stated that for eight months under this treatment she had been free from all morbid symptoms. On the 17th a "vision" occurred for the first time. She saw a red shining ball three times before the left eye, then the usual bright disk. All quickly disappeared with quiet and cold compresses, but on the 18th the patient had a feeling of great weakness, as after an (epileptic) attack though none occurred.

On June 19th 1893 she wrote that by continuing the bromide treatment she had been free from convulsive attacks, while she often has "visions," particularly after excessive use of her eyes,(since the winter of 1890-91).

She differentiates two kinds:

1. Sparks as from an electric machine, which glide along before the left eye as on a spider web and suddenly turn into a shower of sparks. Then a dark spot before both eyes, which gradually becomes violet blue, surrounded by bright stars and disappears after about three minutes.

2. The appearance of the bright disk. It almost always precedes the hemianopsia for ten minutes. Then two glittering points appear, from which a scotoma is developed extending over the whole visual field and is of the most different colors "like a kaleidoscope." While the bright disk occupies the whole visual field vertigo and *violent pain in the head and eyes* occurs, and on the side opposite to the migraine scotoma. These attacks last 25 minutes. They may be shortened by phenacetine 0,5, while antipyrine is ineffectual. In December, 1893, I had occasion to examine the patient again. She had been happily married for four months, free from epileptic seizures under bromide 3,0 daily, had rare attacks of hemicrania ophthalmica gener-

ally during menstruation, first on the right, then on the left, but mostly on the left. But the hemianopsia was always bilateral. Pressure on the left supraorbital nerve still suffices to induce flashes of light.

Case 14. W., 14, school boy, of a perfectly healthy father, but his mother is an extremely nervous woman. His brother had ophthalmic migraine at puberty. A brother of the father had the same.

The patient is an intelligent boy without a sign of degeneration. He has never had convulsions. Since his ninth year the patient has complained of a scotoma appearing periodically before the left eye, which he calls his "sun." It occurs after slight physical or mental exertion, never lasts longer than six minutes and was usually followed by an intense headache on the right side.

The patient describes his scotoma as follows:

A circular disk appears before the left eye alone, which is completely surrounded by rays. This disk presents an upper irregular field of blue, a similar middle one of green and a much larger one of yellow. The rays are silver white and intensely brilliant, like the color fields of the disk. The disk increases in size and finally occupies the whole right visual field. There are occasional moments when all is black before the right eye. The left eye is unaffected.

After the tenth year true epileptic seizures followed very intense attacks of scotoma. He had six of these on the day of consultation, (October 19th, 1895). Since then he has dreaded the appearance of the "sun." He then becomes anxious, excited, disturbed. If the "sun" continues to grow larger he knows well that a convulsive attack impends. He complains that the sun affects his brain and quickly loses consciousness. So long as he takes bromide (4,2 daily) the epileptic seizures are controlled, the attacks of migraine are more rare and usually limited to mere scotoma without headache.

In the intervals the patient is perfectly well. Ophthalmoscopic examination negative. Sodium bromide 3,5 with antipyrine 0,8 *pro die* ordered.

Under this treatment no epileptic seizures and migraine scotoma rare.

Case 15. D., 26, working woman, whose mother and sister have had simple migraine. Since puberty the patient has had attacks of migraine, always pre-menstrual. In the beginning she had scotoma, which is compared to the dancing of dust in a sunbeam. At the same time she sees nothing from her left eye. This scotoma lasts ten minutes. Headache follows it. For two years in about every second attack, which then develops especially intense migraine scotoma, she becomes confused, as at first, tries in vain to articulate, does not respond to questions or contact, lets fall anything she has in her hands, wanders about in a dazed condition with eyes open and pupils dilated. This state lasts as long as the scotoma, *i. e.*, ten minutes. She then comes to herself with headache and does not know in the least what has occurred during this time (psychical equivalent of an epileptic seizure). In this period of the attack she is pale, while at the time of the headache her face is hot and suffused. Five months previously she had the first true epileptic seizure while asleep. When the convulsions were over she had her usual migraine. Since then frequent recurrence of the epileptic seizures, generally at night when asleep, so that nothing is to be ascertained as to the migraine scotoma, yet when she comes out of the seizure she always has hemicrania.

No morbid symptoms in the interval. Under bromide (5,0) the seizures cease.

Case 16. P., 29, mechanic, of neuropathic constitution, father unknown, mother neurotic, who committed suicide, had convulsions when a child.

Since 19 violent attacks of scotoma, generally on awakening, lasting for one minute without other attendant symtoms of hemicrania.

Under bromide treatment these attacks disappeared. Recurred at 18. Again disappeared under bromide.

On suspending the bromide the attacks returned. In 1890 in connection with an attack of scotoma the first epileptic seizure. Now in spite of bromide (10.0 *pro die*) the

epileptic seizures continued, always preceded by scotoma. Instead of this occasionally mere fainting spells.

In 1891 he strangled his mother during a state of post-epileptic excitement. Amnesia. No more epileptic seizures since April 7th, 1895, but frequent scotoma as an aura of genuine attacks of hemicrania, without any disorder of consciousness, and of three hours duration.

On December 25th, 1895, slept well after drinking half a litre of wine. On the morning of the 29th scotoma, followed by a state of epileptic psychical excitement of several hours' duration, without convulsions. Amnesia.

Cranium rachitic. No scars on the head. In February, 1896, two such attacks. Under 10.0 of bromide cessation of these attacks, as well as those of ophthalmic migraine.

The following propositions may be formulated as corrolaries from the above cases and as points of view for further research:

1. There are hemicranias differently qualified diagnostically and prognostically. Two varieties of migraine may occur in the same individual (case 5,9).

2. Hemicrania may have a symptomatic significance, as in organic brain diseases, as well as in the neuroses (epilepsy).

3. Ophthalmic migraine and that combined with sensory Jacksonian very often have a symptomatic significance, almost certainly when the hemicrania is acquired (not inherited) and tardive.

4. The migraine clinically related to epilepsy seems exclusively related to the three categories cited, at any rate no evidence exists, that a simple migraine could play such a role.

5. The external sign of clinical homogeneousness of hemicrania and epilepsy is principally a common visual aura, which may occur as a red color (case 11). This common visual aura is the more worthy of note, as it becomes *qua* migraine scotoma neither the ordinary migraine or *qua* optical aura of simple epilepsy, produces rather special clinical types of both neuroses.

If the conditions for the manifestation of this common

symptom were well known, the insight into the clinical homogeneousness of both neurotic types would be greatly facilitated.

It can not be doubted that this visual aura is a symptom of a clinical entity.

It is possible (case 10), that sensory Jacksonian, quasi as a sensory aura may replace the optical. How this aura (optical, possibly sensory) as a common symptom of two neuroses acquires relations to those permanent brain changes, which we must assume for hemicrania, as well as for epilepsy, is very vague.

Facts indicate that this is the more readily possible, when the hemicrania is associated with symptoms, which indicate a territorial extension in the cortex not occurring otherwise.

6. The visual aura may occur sporadically (abortive attack), bring about an attack of hemicrania or epilepsy, or replace both.

In the latter case the one neurosis cannot be regarded as the agent provocateur of the other; both rather are equivalent to each other, to be referred to a common brain change differing in intensity or extent.

7. The(migranous) epilepsy and the (epileptiform) migraine may replace each other. In the first instance psychical and convulsive attacks are possible as substitutions.

8. Where migraine and epilepsy are clinically related, the latter appears as sensory Jacksonian (cases 5-10) possibly postepileptic psychical seizures (7)., as classical seizures (2,3,4) possibly also as a psychical equivalent (15, 16).

9. Ophthalmic migraine is always suspicious of an epileptic significance, almost certainly when epileptic migraine is to be regarded as that combined with sensory Jacksonian.

Epileptic attacks of any kind with visual aura (11-16) always suspicious relations to migraine, but very particularly unilateral headache with vomiting (13), when they appear only with temporary hemicrania, never in the interval.

10. Hemicrania also needs a differentiation therapeutically according to its etiological forms.

The migraine clinically connected with epilepsy may be benefited by anti-epileptic treatment (13-16). Bromide combined with antipyrine proves especially useful.

Much greater are the difficulties met with when it is attempted to establish the relations between hemicrania and hysteria. Owing to the frequency of both neuroses, particularly in the female sex, that a coincidence of the two may exist, is to be admitted *a priori* and is recognized by every practitioner. Also the coincidence of hemicrania and hysterical seizures in point of time need not seem strange, for emotion may be the agent provocateur of both. French authors (Charcot, Babinski, Fink and others) go even farther, in that they assume a clinical connection between the two neuroses.

The conclusive case for this clinical conception is the following reported by Charcot in his *Lecons du mardi á la Salpetriere*, 1887-88, p. 10:

Prud——, 21, engraver, from *conjunctivitis* had attacks of lacerating pains and dimness of sight, which recurred at the same hour daily.

After emotion at the end of January he had the first attack of hysteria *gravis*. This attack recurred daily at the same time until the middle of February and appeared without *prodromi*.

From then an aura (pain, which extended from the vertex to the left eye, then scotoma, which filled the whole visual field) preceded it by a quarter of an hour.

This aura (migraine scotoma) often occurred without being followed by an attack. Quite often, instead of this visual aura, mutism appeared before the hysterical attack. Under bromide treatment (3-5.0) the hysterical attacks disappeared and the migraine symptoms became milder.

Babinski, who reproduces this case (*Archives de neurologie*, 1890, XX. 60) favors the significance of the hemicranial symptoms in this case, in that they often occur as an aura of a hysterical seizure, further that this hemicranial aura is occasionally represented by a specific hysterical symptom

(mutism). Therefore this equivalent hemicrania must be regarded as hysterical. In support of this assumption, that hemicrania may be a syndrome of hysteria, Babinski presents three more cases, but in which he regards only the first and second of the whole number as evidential.

His second case is a girl of 16, who for three months has had daily simple migraine on the right side, after ten days pains occurred at the sixth dorsal vertebra, in the *supra-* and *infra-*mammary region, five days later local hysterical spasms (*globus*, clonic spasm of the right eyelid) with ophthalmic migraine, when the migraine could be induced from the sixth dorsal vertebra (*point migrainogenie.*)

His third case was a young lady of 22, who had *pavor nocturnus* for three to five years, the first attack of ophthalmic migraine occurred at 16 after emotion, which was followed by another with loss of consciousness and convulsions. Fourteen days later scotoma for one hour followed by hemicranial pain for several hours.

Such attacks af ophthalmic migraine after that recurred about every fourteen days while asleep, and were repeatedly benefited by bromide. Recently (without the bromide) attacks daily, but being restricted to mere scotoma. Pressure over the left ovary produces it experimentally, but feebly each time, the same emotion or also its mere memory, while they were benefited by suggestive treatment.

Babinski's fourth case is a girl of 21. Attacks of hysreria *gravis* for four years, ophthalmic migraine for two months, with whose occurrence the attacks of hysteria have disappeared. These attacks of migraine are induced and relieved *ad libitum* by hypnotic suggestion.

At any rate these cases show a close clinical connection of both neuroses, in so far as the ophthalmic migraine may become an aura of a hysterical seizure, permanently replace such attacks, even by irritation of a hyperaesthetic cutaneous area, but which does not prove to be spasmogenic, in one case even produced by hypnotic suggestion.

In another case, in which a mere memory sufficed to induce migraine scotoma, this is also capable of being caused by pressure on one ovary.

We must always beware of hasty conclusions. That in hysteria, which may imitate everything possible, even organic spinal diseases, migraine may also be induced by psychical influences, that in the very intense and parodoxical susceptibility of the nervous system in such patients a mechanical irritation is able to do this, should not seem strange. However, it is striking, that from the champions of the opinion that migraine may be a syndrome of a hysterical neurosis or equivalent of a hysterical seizure, only thirteen of such cases could be cited until 1891 (see *Gilles de la Tourette traité de l'hystérie*, p. 379), of which the majority are not unobjectionable and only prove a coincidence of ophthalmic migraine and hysterical syndromes.

In view of this fact it must remain an open question, whether migraine may play the same role with respect to hysteria, as it actually does to epilepsy.

Previous observations indicate, that in hysterics psychical and mechanical irritants exceptionally suffice to induce an attack of migraine, and that an attack of migraine may be the agent provacateur of an hysterical seizure, in the way somewhat that the area of hemicranial hyperaesthesia becomes temporarily or permanently a spasmogenic zone, analogous to numerous cases (Schützenberger, Baetian and others), in which another nerve affected with neuralgia acquires this importance. Under all circumstances, and analogous to epilepsy, only ophthalmic migraine could possibly attain the significance of a syndrome or equivalent of the hysterical neurosis, for the certain differentiation of simple migraine from certain cases of *clavus hystericus* with sensory hyperaesthesia and vomiting, as well as from so-called pseudo-meningitis hysterica, is a doubtful fact.

In my experience I find numerous cases of simple and ophthalmic hemicrania in hysterics, but not one, in whom the attack of migraine could be interpreted as a syndrome or equivalent of the hysterical neurosis; whereas I know of several cases, in which the attack of migraine was evidently the agent provocateur for the recurrence of hysterical seizures, whence it may be assumed, that the area of migraine had temporarily taken the role of a hystero-

(*spasmo*) genic zone. Inversely the hysterical attack may provoke the migraine.

The following cases may serve as such examples:

Case 17. Mrs. Z., 35, has had ophthalmic migraine since 30. No instances of this trouble in the family. Her brothers and sisters are extremely neuropathic. Since 25 this extremely nervous woman has had attacks of hysteria *gravis*. These almost exclusively follow emotion and recur at intervals of weeks to months. The hemicrania developed five years ago after pregnancy. It is never sporadic, but always appears as a result of hemicranial attacks, which begin with headache on the left side and last for one to two hours. In more rare instances, the hemicrania develops at the acme of the attacks, apparently from the hysterogenic zone, but usually in connection with the convulsion. The hemicrania is always located on the left side and lasts for two days. Following it, inability to turn the head to the left exists for about fourteen days.

Large, handsome woman. A circumscribed area over the left parietal eminence is hyperalgesic in the intervals and sensitive to pressure, as well as the left ovary. No other stigmata hysteriae.

Case 18. Miss T., 21, from France, governess, of a neuropathic family. Parents and all her brothers and sisters have hemicrania, also one sister has hysteria gravis. Patient very talented, has had no serious illness, but afflicted with typical simple migraine since 15. Since her first menstruation she has had occasional attacks of hysteria gravis, the last one a year ago. Owing to the family's loss of property the patient has recently had to take a position as governess. She became very emotional over this, on October 7th, 1893, had an attack of hysteria gravis (laughed, cried, threw herself about, rolled her eyes, ground her teeth, raved about flowers, her father had become insane, she would also, protests, etc.).

This condition lasted until admitted to the Clinic (October 9th) and ceased on October 10th. Only summary memory. Patient claims her migraine has never been ophthalmic.

Worthy of note are mouches volantes and erythropsia in the fixation of objects. Besides clavus and sensitive ovary, she presents the true hysterical character.

On October 14th, 19th, 20th and 29th, the above described and purely psychical attacks of hysteria gravis occurring as insignificant convulsive symptoms have recurred. Their starting point is evidently the clavus. In the majority of attacks it may be proven that hemicrania preludes and accompanies them. The patient confirms this from her previous experience. The parietal eminence (clavus) is given as the starting point of the hemicrania.

On November 6th, the last attack of hysteria gravis (merely delirium, which was about flowers, but with terrifying hallucinations periodically), of four hours duration with initial migraine and evidently inducing the attack. During the attack of hysteria headache on one side, but without any relation to the delirium. On December 20th, the patient was discharged "recovered".

Case 19. Early July, 19th, 1896, M. K., 22, single, was brought before the magistrate by one of the police, whom she had accosted with the words: "You are my doctor." She seemed confused, excited, expectorated constantly, presented marked change of disposition, complained of violent headache, owing to which she had already been repeatedly in confinement. Owing to great restlessness and fear she had gone to Vienna, looked for some place to drown herself all day, as she no longer enjoyed life. She then began to whistle and sing. Brought to the Clinic, she is still somewhat confused, has only a summary memory of recent events, is fearful, unstable, presents great change in disposition, erotic manner. She complains of headache, presents pressure pain points over the left supraorbital and temporal nerves, left hemihyperaesthesia, concentric contraction of the visual field, otherwise no hysterical stigmata. On the night of the 20th the patient slept well. Menses began on the 20th, with violent colic and excerbation of the headache on the left side, with the attendant fancy that someone had stepped on her head, but which idea is not retained. Patient is no longer

confused, but emotional, fearful, first erotic, then depressed from taedium vitae, the whole picture of marked hysterical stamp. With the cessation of menstruation on the 25th, these symptoms disappeared. Patient became quiet, oriented, appeared mentally dull, somewhat defective ethically and gave the following anamnesis:

Alcoholism, insanity and tuberculosis have often occurred in the family. Her father died of phthisis, one sister committed suicide in an insane asylum, her mother choleric.

Patient has a rachitic cranium and evidence of rachitis elsewhere in the skeleton. She had the usual diseases of childhood, also attacks of hysteria gravis from 9 to 15 from a severe fright. These have occurred again since 18, but rarely.

From 14 to 17 the patient had traveled about with a concert troupe. She returned home and became reconciled with her mother over quarrels about a man whom she should marry against her will. When the patient was nearly 19 she became pregnant and in an abnormal (hysterical?) psychical condition attempted suicide by a solution of phosphorous and by jumping from the window. Since that occurrence attacks of violent headaches on the left side, preceded by seeing black rings before the eyes, still without *mouches volantes;* at their acme nausea.

This hemicranial symptom complex has since been related to attacks of hysteria *gravis*, in so far, according to the patient's statement, the aura of the globus and vertigo have been associated with scotoma and headache.

During the past year the convulsive attacks of the hysterical neurosis have become very rare. But following the above symptoms of aura psychical attacks more often occur in the form of confusion, agitated restlessness, *taedium vitae*. In one of these attacks she jumped into the river. Owing to such attacks the patient has been in the insane asylum twice, has been at times a waitress and mistress. One day her lover was arrested as a swindler (July 7th, 1896.) She fled in fear to her mother at Graz. From then no hemicranial seizures with agitated confusion, taedium

vitae and terrifying sense deceptions became more frequent. She was kept at home. In connection with such a seizure she fled to Vienna (July 17th) to seek employment.

On the 18th hemicrania began, she became confused, was again fearful, had taedium vitae, wanted to jump into the Danube, gave away her effects, as they were useless to one traveling, and wandered about the city. She summarily remembers having tried to pray in several churches, but failed to do so from restlessness and fear. She has also been to a cemetery to see how her future resting place looks. In the evening, with excerbation of the headache, she became more confused and was arrested in this condition.

During the following weeks of observation the patient presented traces of psychical degeneration, as well as those of a hysterical character. Recently attacks of migraine or hysterical delirium were no longer observed.